Relation of oropharyngeal palsy to neck and limb weakness in Guillain-Barré and Fisher's syndromes

	Oropharyngeal palsy				
	Present (n=48)	Absent (n=104)	p Value	Odds ratio	95% CI
Ophthalmoplegia	18 (38%)	35 (34%)	0.6		
Neck weakness	36 (75%)	33 (32%)	<0.0001	6.5	3.1–13.6
Arm dominant weakness	20 (42%)	13 (13%)	<0.0001	5.0	2.3–10.9
Leg dominant weakness	11 (23%)	50 (48%)	0.003	0.3	0.1–0.7

Differences in proportions were examined by χ^2 test. 95%CI=95% confidence interval.

only in patients who have a restricted distribution of muscle weakness in the pharynx, neck, and proximal upper limbs but no weakness or areflexia in the legs. In his original report,[2] however, one of the three patients with PCB had generalised areflexia. Moreover, the patient with Guillain-Barré syndrome described by Mizoguchi *et al*,[3] whose initial symptoms were lower cranial nerve dysfunction and upper limb weakness, later developed generalised muscle weakness. These patients with PCB with generalised areflexia or weakness indicate that the preservation of the tendon reflex and muscle power in the legs depends on the severity of the involvement of the limbs. None of the patients in our study met the clinical criteria proposed by Ropper.[1] However, the close association of weakness of the pharynx, neck, and upper limbs in Guillain-Barré syndrome and Fisher's syndrome indicates that PCB is a distinct variant of Guillain-Barré syndrome, because ophthalmoplegia, a cardinal sign in Fisher's syndrome, is not associated with oropharyngeal palsy, neck weakness, or arm dominant weakness.

Our finding is also supported by detection of serum antibodies against GT1a ganglioside in patients with PCB which show different reactivity from those in patients with Fisher's syndrome.[3 4] IgG anti-GT1a antibodies in patients with PCB are not absorbed by GQ1b ganglioside whereas those in patients with Fisher's syndrome are.[4] Because only GT1a is recognised by serum IgG from the patient who had a restricted distribution of muscle weakness in the pharynx, neck, and proximal upper limbs,[4] we speculate that anti-GT1a and anti-GD1a antibodies respectively contributed to the development of PCB and generalised weakness in the patient described by Mizoguchi *et al*.[3]

MICHIAKI KOGA
NOBUHIRO YUKI
KOICHI HIRATA
Department of Neurology, Dokkyo University School of Medicine, Tochigi, Japan

Correspondence to: Dr Michiaki Koga, Department of Neurology, Dokkyo University School of Medicine, Kitakobayashi 880, Mibu, Shimotsuga, Tochigi 321–0293, Japan. Tel: 0081 282 86 1111 extention 2733 or 2578; fax 0081 10-60-89313; email: koga@dokkyomed.ac.jp

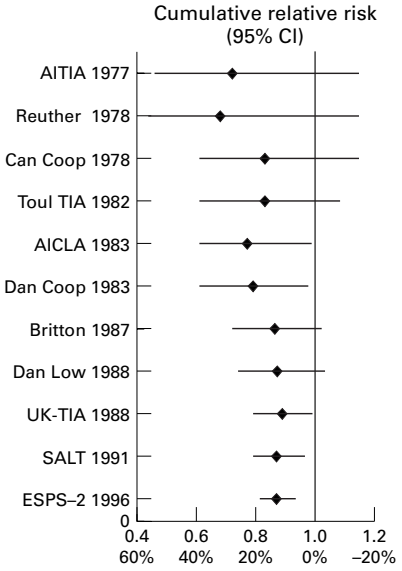
1 Ropper AH, Wijdicks EFM, Truax BT. *Guillain-Barré syndrome*. Philadelphia: FA Davis, 1991:18–21, 73–105.
2 Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. *Arch Neurol* 1986; **43**:1150–2.
3 Mizoguchi K, Hase A, Obi T, *et al*. Two species of antiganglioside antibodies in a patient with a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1994;**57**:1121–3.
4 Koga M, Yuki N, Ariga T, *et al*. Is IgG anti-GT1a antibody associated with pharyngeal-cervical-brachial weakness or oropharyngeal palsy in Guillain-Barré syndrome? *J Neuroimmunol* 1998;86:74–9.

Cumulative meta-analysis of aspirin efficacy after cerebral ischaemia of arterial origin

In 1996 we reported in this *Journal* that there was virtually no difference in relative risk reduction for low (<100 mg/day), medium (300 to 325 mg/day), and high (>900 mg/day) doses of aspirin in the prevention of vascular events in patients with cerebral ischaemia of arterial origin.[1] A meta-analysis of the cumulative data showed a modest 13% (95% confidence interval (95% CI) 4% to 21%) relative risk reduction. Recently the final data of the second European Stroke Prevention Study (ESPS-2) were reported.[2] One of its comparisons was between 50 mg aspirin daily and placebo in patients after cerebral ischaemia; the relative risk reduction of 13% (95% CI 0% to 24%) was exactly the same as that resulting from our previous meta-analysis. This similarity allows the calculation of an update of the meta-analysis. The overall relative risk reduction of course remains 13%, but the 95% CI has narrowed to 6% to 19%. The figure shows the results of the updated cumulative meta-analysis, in chronological order. These data once more underscore the need for more efficacious treatment strategies. For this reason we started the European and Australian Stroke Prevention in Reversible Ischaemia Trial (ESPRIT).[3]

A ALGRA
J VAN GIJN
Julius Center for Patient-Orientated Research, and University Department of Neurology, Utrecht, The Netherlands

Correspondence to: Dr Ale Algra, Julius Center for Patient-Orientated Research, University Hospital Utrecht, P O Box 85500, 3508 GA Utrecht, The Netherlands. Telephone 0031 30 250 9350; fax 0031 30 250 5485; email A.Algra@neuro.azu.nl



Cumulative meta-analysis in chronological order (1977 to 1996) with relative risks and corresponding relative risk reductions with 95% CIs. Each line represents the relative risk and 95% CI of that study combined with all previous studies.

1 Algra A, van Gijn J. Aspirin at any dose above 30 mg offers only modest protection after cerebral ischaemia. *J Neurol Neurosurg Psychiatry* 1996;**60**:197–9.
2 The ESPS-2 Group. European Stroke Prevention Study 2. Efficacy and safety data. Secondary endpoints. *J Neurol Sci* 1997;**151**(suppl): S27–37.
3 Major ongoing stroke trials. *Stroke* 1998;**29**: 1268.(Updated every 4 months.)

CORRESPONDENCE

Hemifacial spasm

We have looked with interest at the scan of a patient with hemifacial spasm by Reigosa and Rios.[1] Indeed, this is a very nice MRI which shows an arterial loop and the internal auditory meatus. However, this loop is not the cause of hemifacial spasm.

Typical hemifacial spasm, which begins in the orbicularis oculi and gradually progresses down the face, is caused by a blood vessel on the non-fascicular portion of the facial nerve on the caudal or anterior aspect, including the intrapontine nerve. Atypical hemifacial spasm, which starts in the buccal muscles and progresses up the face, is caused by a blood vessel on the posterior or rostral side of the nerve. This is much less common. The compression is also at the brainstem. A distal artery, as shown in the scan, does not cause hemifacial spasm. The syllogism that Reigosa and Rios bring out—namely, that botulinum toxin helped and that this picture showed the pathology, is inadequate. They do not have a completed explanation.

This patient's spasm will recur because the cause has not been treated. The spasm has an excellent chance of responding to a microvascular decompression of the facial nerve performed by a neurosurgeon who has experience in the nuances of the operative procedure.

Nevertheless, Reigosa and Rios have shown a beautiful scan.

PETER J JANNETTA
AMIN KASSAM
Department of Neurological Surgery, School of Medicine, University of Pittsburgh, PA, USA

1 Reigosa RP, Rios JP. Hemifacial spasm. *J Neurol Neurosurg Psychiatry* 1998;**64**:687.

Pego Reigosa replies:

We thank Jannetta and Kassam for their interest in our article.[1] We think that the vascular loop that appears in the MR image is indeed the cause of the hemifacial spasm of our patient, as it is the only abnormal finding of the neuroimaging studies performed. Furthermore, we did not find compression of the nerve at other levels where it is more often encountered, as is the caudal aspect of the VII cranial nerve next to the pons.

Moreover, it is evident that the hemifacial spasm will reappear or recur. For this reason,